Amendments to the Claims:

This listing of claims will replace all prior versions, and listings, of claims in the application:

Listing of Claims:

1. (Currently Amended) A method of inhibiting vascular hyperpermeability in an individual comprising the step of administering to said individual a therapeutically effective amount of a compound that selectively inhibits the cellular signaling function of KDR by disrupting the catalytic kinase response of KDR/VEGFR-2 without significantly affecting the activity of Flt-1/VEGFR-1 or other kinases.

2. (Original) The method of Claim 1 wherein said inhibition of the cellular signaling function of KDR is selective for the KDR signaling function.

3. (Original) The method of Claim 1 wherein said cellular signaling function of KDR is stimulated by the binding of an activating ligand to the receptor portion of KDR.

4. (Original) The method of Claim 3 wherein said inhibition of the cellular signaling function of KDR is selective for the KDR signaling function.

5. (Original) The method of Claim 1 wherein said inhibition of the cellular signaling function of KDR is a process selected from the group consisting of blocking the production of an activating ligand, modulating the binding of the activating ligand to the KDR tyrosine kinase receptor, disrupting the dimerization of the receptor, blocking KDR trans-phosphorylation, inhibiting the activity of the KDR tyrosine kinase, impairing the recruitment of intracellular substrates of KDR, and interrupting the downstream signaling initiated by the phosphorylation activity of the KDR tyrosine kinase.

6. (Original) The method of Claim 5 wherein said inhibition of the cellular signaling function of KDR is selective for the KDR signaling function.

7. (Original) The method of Claim 1 wherein said compound inhibits the catalytic kinase activity of said KDR.

8. (Original) The method of Claim 1 wherein said compound is an antagonist of KDR tyrosine kinase activation.

9. (Original) The method of Claim 1 wherein said compound selectively inhibits the phosphorylation of KDR kinase substrates.

10. (Original) The method of Claim 1 wherein said compound is selective for said KDR tyrosine kinase.

11. (Previously Amended) The method of Claim 10 wherein said compound is an organic molecule wherein said compound binds to said KDR tyrosine kinase.

12. (Original) The method of Claim 11 wherein the administration of said compound inhibits the formation of a disease state selected from the group consisting of macular edema, aphakic/pseudoaphakic cystoid macular edema, retinoblastoma, ocular ischemia, ocular inflammatory disease or infection, choroidal melanoma, edematous side-effects induced by iron chelation therapy, pulmonary edema, myocardial infarction, rheumatoid diseases, anaphylaxis, tissue edema at sites of trauma and allergic inflammation, allergies, hypersensitive reactions, polyp edema at sites of chronic inflammation, cerebral edema, brain tumor fluid-filled cysts, communicating hydrocephalus, carpal tunnel syndrome, organ damage resulting from a burn, inhalation burn injury, skin burns, blistering associated with sunburn, irritation or infection, erythema multiforme, edematous macules and other skin disorders, brain tumors, tumor effusions, lung or breast carcinomas, ascites, pleural effusions, pericardial effusions, high altitude "sickness", radio anaphylaxis, radiodermatitis, glaucoma, conjunctivitis, choroidal melanoma, adult respiratory distress syndrome, asthma, bronchitis, ovarian hyperstimulation syndrome, polycystic ovary syndrome, menstrual swelling, menstrual cramps, stroke, head trauma, cerebral infarct or occlusion, hypotension,

ulcerations, sprains, fractures, effusions associated with synovitis, diabetic complications, hyperviscosity syndrome, liver cirrhosis, microalbuminuria, proteinuria, oliguria, electrolyte imbalance, nephrotic syndrome, exudates, fibroses, keloid, and the administration of growth factors.

13. (Original) The method of Claim 10 wherein adverse effects associated with an alteration in the cellular signaling function of tyrosine kinases other than KDR are avoided when said compound is administered.

14. (Cancelled) The method of Claim 1 wherein said compound is selected from the group consisting of single-chain antibodies, KDR-specific ribozymes, and anti-sense polynucleotides, wherein said compound is introduced or produced intracellularly thereby inhibiting the proper presentation of functional KDR tyrosine kinases.

15. (Original) The method of Claim 1 wherein said compound is administered in combination with a pharmaceutical agent selected from the group consisting of an anti-endemic steroid, a Ras inhibitor, anti-TNF agents, anti-IL1 agents, an antihistamine, a PAG-antagonist, a COX-1 inhibitor, a COX-2 inhibitor, a NO synthase inhibitor, a nonsteroidal anti-inflammatory agent (NSAID), a PKC inhibitor and a PI3 kinase inhibitor.

16. (Previously Amended) A method of inhibiting a physiological process or state in an individual, said physiological process or state selected from the group consisting of edema formation, diapedesis, extravasation, effusion, exudation, ascites formation, matrix deposition and vascular hypotension, wherein said inhibiting comprises the administration of a therapeutically effective amount of a compound that inhibits the cellular signaling function of KDR.

17. (Original) The method of Claim 16 wherein said compound is selective for said KDR tyrosine kinase.

18. (Previously Amended) The method of Claim 17 wherein said compound is an organic molecule wherein said compound binds to said KDR tyrosine kinase.

19. (Original) The method of Claim 18 wherein the administration of said compound inhibits the formation of a disease state selected from the group consisting of macular edema, aphakic/pseudoaphakic cystoid macular edema, retinoblastoma, ocular ischemia, ocular inflammatory disease or infection, choroidal melanoma, edematous side-effects induced by iron chelation therapy, pulmonary edema, myocardial infarction, rheumatoid diseases, anaphylaxis, tissue edema at sites of trauma and allergic inflammation, allergies, hypersensitive reactions, polyp edema at sites of chronic inflammation, cerebral edema, brain tumor fluid-filled cysts, communicating hydrocephalus, carpal tunnel syndrome, organ damage resulting from a burn, inhalation burn injury, skin burns, blistering associated with sunburn, irritation or infection, erythema multiforme, edematous macules and other skin disorders, brain tumors, tumor effusions, lung or breast carcinomas, ascites, pleural effusions, pericardial effusions, high altitude "sickness", radio anaphylaxis, radiodermatitis, glaucoma, conjunctivitis, choroidal melanoma, adult respiratory distress syndrome, asthma, bronchitis, ovarian hyperstimulation syndrome, polycystic ovary syndrome, menstrual swelling, menstrual cramps, stroke, head trauma, cerebral infarct or occlusion, hypotension, ulcerations, sprains, fractures, effusions associated with synovitis, diabetic complications, hyperviscosity syndrome, liver cirrhosis, micro alburninuria, proteinuria, oliguria, electrolyte imbalance, nephrotic syndrome, exudates, fibroses, keloid, and the administration of growth factors.

20. (Original) The method of Claim 16 wherein said compound inhibits the catalytic kinase activity of said KDR.

21. (Original) The method of Claim 16 wherein said compound is an antagonist of KDR tyrosine kinase activation.

22. (Original) The method of Claim 16 wherein said compound selectively inhibits the phosphorylation of KDR kinase substrates.

23. (Original) The method of Claim 16 wherein said compound is selective for said KDR tyrosine kinase.

24. (Original) The method of Claim 16 wherein said cellular signaling function of KDR is stimulated by the binding of an activating ligand to the receptor portion of KDR.

25. (Original) The method of Claim 24 wherein said compound is selective for said KDR tyrosine kinase.

26. (Cancelled) The method of Claim 16 wherein said compound is selected from the group consisting of single-chain antibodies, KDR-specific ribozymes and anti- sense polynucletodies, wherein said compound is introduced or produced intracellularly thereby inhibiting the proper presentation of functional KDR tyrosine kinase.

27. (Original) The method of Claim 16 wherein said inhibition of the cellular signaling function of KDR is a process selected from the group consisting of blocking the production of an activating ligand, modulating the binding of the activating ligand to the KDR tyrosine kinase receptor, disrupting the dimerization. of the receptor, blocking KDR trans-phosphorylation, inhibiting the activity of the KDR tyrosine kinase, impairing the recruitment of intracellular substrates of KDR, and interrupting the downstream signaling initiated by the phosphorylation activity of the KDR tyrosine kinase.

28. (Original) The method of Claim 17 wherein adverse effects associated with an alteration in the cellular signaling function of tyrosine kinases other than KDR are avoided when said compound is administered.

29. (Original) The method of Claim 16 wherein said compound is administered in combination with a pharmaceutical agent selected from the group consisting of an anti- endemic steroid, a Ras inhibitor, anti-TNF agents, anti-ILl agents, an antihistamine, a PAF-antagonist, a COX-1 inhibitor, a COX-2 inhibitor, a NO synthase inhibitor, a nonsteroidal anti-inflammatoiy agent (NSAM), a PKC inhibitor and a P13 kinase inhibitor.